# Aseptic meningitis and herpes simplex proctitis

# A case report

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SUMMARY A male homosexual patient developed an aseptic meningitis during the course of acute ano-proctitis due to herpes simplex virus type 2 (HSV-2). Aseptic meningitis (sometimes proved to be due to HSV-2) has been associated with genital but not with anal herpes. This sexually transmissible virus should be considered when patients with aseptic meningitis are investigated.

#### Introduction

The reported incidence of genital infection with the herpes simplex virus (HSV) seems to be increasing. Various neurological syndromes have been described in association with genital herpes. We report a patient who developed an aseptic meningitis during the course of acute herpetic ano-proctitis. This complication of herpes proctitis, although suspected, has not been reported previously.

#### Case report

A 23-year-old man attended the Praed Street Clinic, St Mary's Hospital, London, on 16 June 1980 with a "sensation of swelling in the back" for one day. He had had casual passive homosexual contact four days previously.

INITIAL INVESTIGATIONS AND TREATMENT On examination he had an inflamed rectal mucosa and purulent discharge. Routine investigations for sexually transmitted disease were performed. Microscopy showed polymorphonuclear cells in smears from the discharge containing intracellular Gramnegative diplococci. Ampicillin 3.5 g and probenecid 1 g were, therefore, given in a single dose. Culture results were negative for gonococci.

Two days later he reattended complaining of increasing anal discomfort, pain on defecation, some hesitancy of micturition, and hyperaesthesia in the back of his thighs. On examination there was bilateral tender enlargement of the inguinal lymph

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Accepted for publication 8 July 1981

nodes. Multiple clusters of herpetiform vesicles were seen around the anus. Culture of samples from these vesicles later grew the herpes simplex virus (untyped). He was given co-trimoxazole tablets two twice daily for one week and an analgesic. He did not give any previous history of herpes infection.

#### **COURSE OF ILLNESS**

On 21 June 1980 the patient was admitted to the Royal Free Hospital with symptoms of malaise, headache, photophobia, hesitancy of micturition, and pain radiating down his legs. His temperature was 38°C, pulse rate 93/min, and he had signs of meningism. There was acute localised tenderness over the region of a lumbosacral spina bifida occulta, which he was known to have, but there was no neurological deficit. The perianal lesions had healed, but proctoscopy showed an inflamed ulcerated mucosa.

### LABORATORY INVESTIGATIONS

Lumbar puncture was performed and showed the following results: cerebrospinal fluid clear, pressure 160 mm; WBC  $560 \times 10^6/1$  ( $560/\text{mm}^3$ ) (90% lymphocytes, 10% leucocytes); RBC  $100 \times 10^6/1$  ( $100/\text{mm}^3$ ); glucose 1.5 mmol/l (blood 6.5 mmol/l); and protein 0.7 g/l (normal value 0.4 g/l).

Cultures were bacteriologically sterile, but the CSF was not cultured for herpes virus. Full blood count and differential count, electroencephalogram, and radiographs of the chest were normal. Repeated Venereal Disease Research Laboratory and Treponema pallidum haemagglutination tests gave negative results.

#### VIRAL STUDIES

Rectal swabs taken on 26 June grew herpex simplex virus type 2. Bacterial and viral cultures of throat

swabs gave negative results. Serum antibody titres for cytomegalovirus, mumps S, and mumps V were very low.

Serum herpes virus complement-fixing antibody titres were 1/64 on 26 June 1980 and on 22 July 1980.

#### **MANAGEMENT**

The patient was kept under observation and managed conservatively. His temperature returned to normal on the second day. He made an uneventful recovery and was discharged home after six days. He was seen as an outpatient four weeks later and there was no evidence of recurrence.

#### Discussion

Three days before admission this patient had symptoms suggestive of sensory root involvement. He then developed an aseptic lymphocytic meningitis during the course of active herpetic ano-proctitis. HSV was recovered from ano-rectal swabs both before the onset (untyped) and during recovery (type 2) from meningitis.

HSV accounts for only a small proportion of cases of aseptic meningitis. Active genital herpes—usually a primary attack—coexists with only about one-third of the reported cases of herpes meningitis. Sexual transmission of HSV is known to occur in the absence of signs and symptoms of genital infection. Furthermore, the virus can be recovered from the genital tract or rectum of such latent cases. A history of genital herpes should, therefore, be sought during investigation of the cause of aseptic meningitis. In the absence of such a history or other

obvious aetiology the examination of appropriate ano-genital specimens (including prostatic fluid) for culture of HSV can be helpful, especially since a rise in serum herpes antibody titre may not appear during a recurrent attack.

Apart from epidemiological and theoretical considerations, the diagnosis of HSV as a cause of meningitis could have therapeutic implications in the near future should current evaluations of systemic acycloguanosine in the treatment of herpetic infections prove successful.

We thank Dr J W R Harris, Professor N McIntyre, and Dr D C McDonald Burns for encouragement and advice.

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